Abstract

Guillain-Barre Syndrome (GBS) is an immune-mediated polyneuropathy commonly occurring worldwide and classically appearing as acute flaccid paralysis with ascending limb weakness and reduced or absent tendon reflexes (1). It is characterized by global areflexia and albuminocytologic dissociation in cerebrospinal fluid (CSF). Apart from the classic variants of acute immune-mediated demyelinating polyneuropathy, there are several other rare variants like acute axonal neuropathy, pure motor or sensory neuropathy, Bickerstaff brainstem encephalitis, Miller Fisher syndrome (MFS), Pharyngeal – cervical – brachial variant (PCB), Polyneuritis cranialis, paraparesis and facial diplegia (1). Acute bulbar palsy plus syndrome (ABPp) is an even rarer variant of GBS, which presents as cranial nerve involvement without neck or limb muscle involvement (2). Some of these are discrete illnesses, while some overlap with each other. Here, we present one of the rarer variants of GBS – ABPp. There has been no similar case reported from Sri Lanka so far.

Case Presentation

A 37-year-old previously well male presented with dysphagia, nasal speech and unsteadiness of gait for five days, followed by diplopia and left-sided facial weakness. Onexamination, he was conscious. He showed complex ophthalmoplegia, left-sided lower motor neuron type facial nerve palsy and later bilateral lower motor neuron type facial nerve palsies. Additionally, there were glossopharyngeal and vagalnerve palsies, nasal speech and globalareflexia. There were no other cerebellar signs except mild ataxia and dysarthria. There was no reduced neck muscle power, reduced shoulder muscle power or limb muscle weakness. There was no respiratory muscle involvement. Sensory examination was normal. The nerve conduction study (NCS) showed F wave abnormalities and supported a variant of GBS. Lumbar puncture (LP), which was performed on the 11thday ofillness, revealed cyto protein dissociation with a protein count of 110mg/dL. The diagnosis was made as a GBS variant–acute bulbar palsy plus syndrome, on thebasis of typical clinical presentation, NCS and LP findings with the exclusion of other differential diagnoses. Intravenous Immunoglobulin continued for five days, which resulted in significant improvement of his signs and symptoms over 10-12 days.

Conclusion

Acute bulbar palsy plus syndrome is a rarer variant of GBS, which commonly manifests with bulbar palsy, facial nerve palsy, ophthalmoplegia and ataxia without neck muscle or limb weakness. It should be diagnosed early, as prompt monitoring and treatment is essential to minimize mortality and complications.